Quantifying Muscle Co-activation for Impaired Finger Independence in Stroke Survivors

Yuwen Ruan, Henry Shin, and Xiaogang Hu

***Abstract*— *Objective:* Hand impairment frequently occurs in individuals following a stroke. There is evidence of abnormal muscle co-activation that contributes to impaired control of finger independence. This study quantitatively analyzed hand muscle co-activation patterns of chronic stroke survivors. Systematically quantifying the degree of muscle co-activation patterns in stroke survivors can help us to better understand the mechanisms behind compromised finger independence and enables a more accurate assessment of hand impairment. *Methods:* We analyzed muscle co-activation patterns both macroscopically and microscopically using high-density surface electromyographic (HD-sEMG) signals and decomposed motor unit signals from extrinsic and intrinsic flexor/extensor muscles. The muscle co-activation patterns between both sides of stroke survivors and neurologically intact controls were compared. *Results:* We observed increased levels of co-activation in the affected sides of stroke survivors compared with their contralateral sides and the control groups, with a higher degree in the extrinsic muscles than the intrinsic muscles. The asymmetry in muscle co-activation between hands correlated with impaired finger force independence and clinical assessment scales. In the micro-level analysis of motor unit action potentials (MUAPs) distributions, we observed a notable increase in action potential spread of MUAPs in the individual affected extrinsic muscles, but the altered MUAP distribution did not correlate with clinical assessment scales. *Conclusion:* We systematically quantified abnormal muscle co-activation patterns in impaired finger independence after stroke. *Significance:* With further development, the outcomes provide a comprehensive understanding of hand dexterity deficits in stroke survivors, which may provide guidance for targeted rehabilitation strategies and offer a potential for automated impairment evaluations.**

***Index Terms*—finger independent control, hand impairment, electromyography, muscle co-activation, stroke**

I. Introduction

CEREBRAL stroke is a prevalent neurological condition. According to data collected by the National Health and Nutrition Examination Survey (NHANES) from 2017 to 2020, approximately 9.4 million Americans suffered a stroke [1]. Strokes invariably exert significant negative impacts on their daily lives [2], [3]. Notably, around two-thirds of stroke survivors endure persistent deficits in hand function [4], [5], and the recovery of hand dexterity is deemed important yet challenging in the rehabilitation process. Hence, understanding the mechanism of impairment and accurately evaluating impaired hand function are vital for enhancing quality of life.

In individuals without neurological impairments, our central nervous system can coordinate muscle activation patterns with high selectivity depending on the task requirements. This selectivity is achieved through the convergent and divergent neuronal projections to the spinal motoneurons and interneurons. However, in stroke survivors, the impairment of corticospinal projections increases their reliance on remaining undamaged descending pathways. This, in turn, could lead to abnormal co-activation of muscles [6]–[8], which refers to abnormal simultaneous activation of multiple muscle groups or muscle compartments. For example, a prior study [8] has suggested that the remaining intact cortical areas and pathways in stroke survivors might offer compensatory coordination of muscle activation, but with reduced selectivity. This abnormal pattern of muscle activation results in the inability to achieve independent finger control, which is a common manifestation of hand impairment following a stroke. The extrinsic finger muscle groups are unique multi-compartment and multi-tendonous muscles. The complex anatomy largely precludes accurate recordings of the activation of individual compartments using traditional surface electrodes due to inevitable crosstalk [9]. Furthermore, there are limited studies regarding the size and anatomical organization of the intrinsic muscles post-stroke, and the relative contributions of intrinsic and extrinsic muscles to impaired finger flexion or extension remain unclear [10].

Currently, the evaluation of impairments in stroke patients heavily relies on standardized clinical assessments. During these assessments, stroke survivors are instructed to perform a variety of fundamental movements, and clinicians gauge their motor function by assigning scores based on their performance. The Action Research Arm Test (ARAT) and the Chedoke-McMaster Stroke Assessment are two of the commonly utilized assessments for hand function in stroke survivors [11]. The ARAT comprises 19 items grouped into four subsections: grasp, grip, pinch, and gross arm movement. Each item's performance is rated on a 4-point scale, ranging from 0 (no movement possible) to 3 (movement performed normally) [12]. The Chedoke-McMaster Stroke Assessment evaluates six dimensions: shoulder pain, postural control, the arm, the hand, the leg, and the foot. Each dimension is measured on a 7-point scale, with higher values signifying better motor status. [13] Stroke survivors typically undergo

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these tests during their routine clinic visits, with clinicians using the obtained scores as a foundation for determining the subsequent steps in their treatment plan.

Despite the widespread utilization of standardized clinical assessments, this evaluation method continues to possess certain limitations. Firstly, the process of assessment through standardized clinical evaluations is inherently subjective and intermittent. The assignment of scores relies on clinicians and is based solely on a single instance of hand movements, resulting in a subjective and potentially inaccurate evaluation. Multiple factors can influence the ultimate scores, including the patients' level of concentration during the tests, their familiarity with the assessment items, and the variability in scores assigned by different clinicians for the same performance. Secondly, clinical assessments are typically conducted in a controlled clinical setting, which diverges from the real-world environment of daily life. In everyday life, individuals encounter unpredictable environmental obstacles and distractions that are not taken into consideration during clinical assessments. Consequently, to enhance the effectiveness of the rehabilitation process, there is a pressing need for a more objective and accurate assessment.

To evaluate hand function objectively and continuously in stroke survivors, it is crucial to quantify the degree of hand impairment. We hypothesize that the level of muscle co-activation serves as an indicator of finger independent control, thereby facilitating the measurement of hand impairment. An earlier work quantified muscle co-activation patterns at the elbow and shoulder in individuals with hemiparetic strokes [6]. In our prior investigation [14], we successfully demonstrated the feasibility of utilizing high-density surface electromyographic (HD-sEMG) signals to assess co-activation in extrinsic muscles. The findings revealed a correlation between the muscle co-activation pattern and finger independence as well as clinical assessment scales for hand impairment. Accordingly, assessing and quantifying the degree of muscle co-activation during finger movements in stroke survivors may offer a more precise, objective, and continuous evaluation of hand function impairment.

In this study, we evaluated the relation between abnormal co-activation patterns and hand function impairment in stroke survivors. We gathered HD-sEMG signals from both extrinsic and intrinsic muscles during finger movements in both stroke survivors and neurologically intact controls. To assess the degree of muscle co-activation, we employed the 2D cross-correlation coefficient of energy maps between individual finger movements and four-finger movements. Principal component analysis (PCA) was utilized to evaluate finger force independence. Subsequently, motor unit decomposition of HD-sEMG was performed to measure the spatial spread of action potentials from individual motor units. Our findings indicate a significant increase in the correlation coefficient and the spread of action potentials on the affected sides of stroke survivors compared to the contralateral sides and intact controls. The observed abnormal muscle co-activation demonstrates a strong correlation with finger force independence and clinical assessment scales, which suggests that the proposed quantification method for abnormal co-activation could serve as an effective assessment for hand function impairment in stroke survivors.

II. Methods

A. Paticipants

We recruited 12 stroke subjects with the following inclusion criteria: (1) Individuals with a single hemispheric stroke incurred at least 6 months prior to enrollment; (2) Unilateral impairment of hand function (Stage of Hand 2-6 on the Chedoke-McMaster Stroke Assessment); (3) No marked increase in muscle spasticity (modified Ashworth scale < 2), and muscle tone with resting flexion force at neutral position < 20 N; (4) Passive range of motion to at least a neutral position; (5) No hand deficits prior to the stroke. (6) Ability to provide informed consent; (7) Medically stable: No concurrent severe medical illness; (8) No upper extremity pain, inflammation, or recent injury; (9) No history of multiple or recurrent vascular episodes. We also recruited 12 neurologically intact control subjects (age-matched with the stroke cohort). All the stroke survivors and intact controls are right-handed. All participants received and signed consent forms with the study protocol approved by our local institutional review board.

B. Experimental Protocol

Participants were seated upright in a chair with their forearm in neutral position resting on a table and wrist in 0° (radial/ ulnar) deviation. The distal and intermediate phalanges of individual fingers were attached to load cells (SM-100, Interface, Inc) through a finger strap, and the load cells, measuring each finger flexion/extension forces, were attached to a custom-made holder fixed to the table (Fig. 1). A U-shaped wooden block fixed to the table was placed to the palmer and dorsal sides of the hand with form padding to reduce force contamination from the wrist. To better illustrate the EMG electrodes on the hand, the U-shaped block is not shown in Fig. 1. The force signals were amplified and sampled at 1 kHz. Two HD-sEMG grids (each with 8x16 channels, with 3 mm diameter recording electrodes and a 10 mm inter-electrode spacing) were placed over the anterior and posterior sides of the forearm to measure extrinsic finger muscles based on multiple bony landmarks (Fig. 1). To facilitate electrode grid placement for the flexor digitorum superficialis (FDS) muscle, an ultrasound scan (Sonoscape S2) was first performed to identify the anatomical distributions of the muscle. Additionally, two grids (8x4 channel) were placed on the dorsal and palmar sides

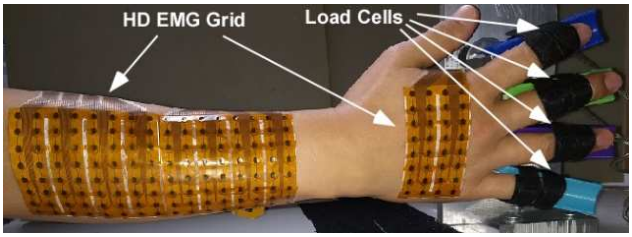

Fig. 1: EMG and finger force setup. The fingers are fixed to the load cells using Velcro straps.

of the hand to record the intrinsic finger muscle activities. The monopolar EMG signals were amplified with a gain of 1000 at a bandwidth of 10-900 Hz and were sampled at 2048 Hz using the EMG-USB2+ acquisition system (OT Bioelettronia, Inc). We made an effort to have consistent electrode placement between arms of the same subject.

Both groups performed the same tasks using both their hands sequentially in two separate sessions. The hand testing order was randomized across subjects, to balance any possible order effect. Prior to the testing, subjects were asked to perform maximal voluntary contractions (MVCs) for 3 s by flexing/extending one or all their fingers isometrically. As the MVC of individual fingers tends to be lower when all fingers are activated concurrently[15], [16], the MVC was calculated either from a single load cell in the case of individual finger flexion/extension conditions, or from the sum of all 4-finger flexion/extension forces.

The main experimental protocol consisted of a series of isometric voluntary contractions, during which the subject was asked to track trapezoidal force trajectories displayed on a computer screen. Peak force amplitudes for the trapezoid were set to percentages of the MVC. The forces on the instructed fingers were displayed, but all the four finger forces were recorded for later analysis. Two steady state force levels (20% and 50% MVC) were tested in random order. The steady state contraction of 8 s was used. During the experiment, subjects were asked to flex/extend their individual finger isometrically against the load cells, while minimizing the forces of other fingers, and these trials were termed 'single-finger tasks'. The subjects also flex/extend all their four fingers simultaneously, which were termed 'all-finger tasks'. In all the tested conditions, the subjects were instructed to minimize wrist motion, and they were asked to repeat the movement when wrist motion was observed, or wrist muscle activation was evident from the EMG map. The subjects repeated the same task 5 times with a 60-s rest period between contractions, and if necessary, longer resting time was provided to minimize fatigue.

Clinical assessments: Clinical assessments were performed on the recruited stroke participants by an occupational therapist. The functional assessments included the Action Research Arm Test (ARAT), and the motor impairment assessments included the hand component of the Chedoke-McMaster Assessment.

C. Data Analysis

Muscle co-activation: We first quantified the degree of muscle co-activation. The EMG signals during the 8 s steady-state hold period were analyzed. Prior to the analysis, potential motion artifact and power line noise were removed with minimal distortion to the EMG signals [17]. The sum-of-squared values of the monopolar EMG of each channel were calculated as the energy of the EMG. The average of the five repetitions were calculated for each channel. Then, the 2D energy map was calculated based on the EMG channel distribution to capture the spatial patterns of muscle activation. Each constructed map was normalized such that the values at each map ranged from 0 to 1. The 2D cross-correlation coefficient of the energy maps between the individual finger tasks and the four-finger task was calculated for each individual muscle using Equation 1 to quantify the degree of muscle co-activation patterns.

$$r_{2D} = \frac{\sum_m \sum_n (A_{mn} - \bar{A})(B_{mn} - \bar{B})}{\sqrt{(\sum_m \sum_n (A_{mn} - \bar{A})^2)(\sum_m \sum_n (B_{mn} - \bar{B})^2)}} \quad (1)$$

where matrices A and B have m x n dimension (e.g., the extrinsic muscle energy map has an 8 x 16 dimension). $\bar{A}$ and $\bar{B}$ are the grand mean of matrix A and B, respectively. A high 2D correlation coefficient signifies that the energy map during individual finger tasks is similar to the energy map during all-finger task. Therefore, a higher correlation signifies that there is substantial muscle co-activation in single-finger tasks similar to that in the all-finger task, thus indicating reduced finger individualization.

To identify potential associations between the muscle co-activation patterns and the force deficits and/or clinical outcomes, the altered activation patterns of each muscle will also be quantified by the asymmetry of correlation measurement between the affected and contralateral sides of each stroke subject, defined by Equation 2.

$$Correlation\ Asymmetry = \frac{Corr_{contra} - Corr_{affect}}{Corr_{contra} + Corr_{affect}} \quad (2)$$

where $Corr_{contra}$ and $Corr_{affect}$ are the correlation coefficients of the contralateral and affected sides, respectively.

Finger force independence: We then evaluated the degree of independent finger force signifying hand dexterity by calculating the dimensionality of the extension/flexion forces during the ramp-up and ramp-down phases, using PCA [18]. We expected that the impaired hand would have a low dimensionality, i.e., a highly correlated finger force output with limited hand dexterity. The difference of variance accounted for between the first PC (with the highest variance accounted for) and the remaining three PCs was calculated, and the average of the difference was used as an indicator of finger independence. A higher difference indicated a smaller degree of finger independence.

Motor unit action potential distribution: We first performed motor unit decomposition of the HD-sEMG signals from the extrinsic muscles using previously developed blind source separation algorithms [19]–[21]. We only focused on the extrinsic muscles, because the intrinsic finger muscles are small, and any reinnervation may not be captured by changes in action potential distribution. The action potential shapes from each channel were calculated using a spike triggered averaging technique [22], [23], which is a system identification method that can extract action potentials based on discharge timings while attenuating background noise and non-time-locked information.

We then determined the spatial distribution of motor unit action potentials. The monopolar action potentials were used to determine 'passive' and 'active' channels. Specifically, if the peak-to-peak amplitude within ±10 ms of the spike timing for a channel was larger than 3 x SD of the baseline (outside of the ± 10 ms window), the channel was noted as 'active'. Otherwise, the channel was considered primarily baseline noise and was noted as 'passive'. This approach had been validated previously [24]. Fig. 2 shows exemplar action potential distributions of

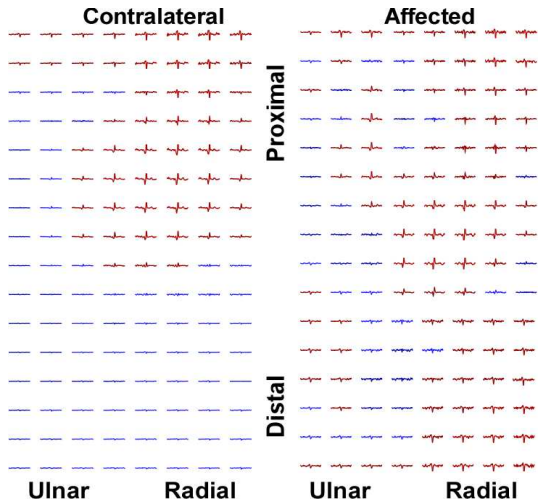

Fig. 2: Monopolar action potential distribution of motor units. Active channels with substantial amplitude are in red, and passive channels with only baseline noise are in blue.

two motor units during middle finger extension of a stroke survivor. The action potentials of a single motor unit in the contralateral muscle are localized to a well-defined region. In contrast, the action potentials of the motor unit in the affected arm are distributed sporadically, indicating neuronal reinnervation. There is evidence of motoneuron loss after stroke and subsequent neuron reinnervation of the previously denervated muscles. The neuron reinnervation can lead to altered MUAP distributions. To quantify the spatial spread of the action potentials of individual motor units, we calculated the variability (standard deviation (SD)) of the active channel spatial location. A higher variability signified more widely distributed action potentials over the muscle.

D. Statistical Analysis

The 2D correlation measures (Equation 1) were tested using paired t-tests for bilateral comparisons in stroke survivors. Independent t-tests were performed between the intact controls and each side of stroke survivors. Because the correlation ranges from 0 to 1, a z-transformation was performed on the correlation coefficient values prior to statistical evaluations. We then performed a linear regression between the force independence deficits (quantified by PCA) and the abnormal muscle co-activation patterns of different finger muscles (asymmetry index of 2D correlation measures in Equation 2), which provided information regarding the contribution of specific muscle impairment to reduced hand dexterity. Because the correlation asymmetry values range from -1 to 1, a z-transformation was performed, and the normality of the regression residual was evaluated to ensure that the regression was valid. We also evaluated potential associations between abnormal muscle co-activation and the clinical assessment scores of stroke survivors. Lastly, spatial distribution of action potential (variability of active channels) was evaluated using paired t-tests for bilateral comparisons in stroke survivors and independent t-tests between controls and each side of stroke survivors. The Bonferroni correction was used to compensate for increased type I error in multiple t-tests.

III. Results

A. Muscle Co-activation Patterns

We first quantified the muscle activation patterns in intrinsic and extrinsic finger muscles during voluntary effort. The 2D energy map across individual channels was then used to capture the spatial patterns of muscle activation. Fig. 3 shows examples of 2D energy maps of the extrinsic extensor (8x16 channel) and dorsal intrinsic (8x4 channel) finger muscles in a stroke survivor with moderate hand impairment (hand component of Chedoke=4 out of 7). The activation patterns on the contralateral arm (bottom row) exhibited distinct localized activation across different tasks (i.e., generation of forces with different fingers); this distinction was especially prominent in the intrinsic muscles. In contrast, the activation patterns on the affected side (top row) tended to show widespread activation with less distinction between patterns across different tasks.

To quantify the degree of muscle co-activation, we calculated the 2D cross-correlation coefficient of the energy maps between the single-finger and the four-finger tasks for each individual muscle to quantify the degree of muscle co-activation patterns in the single-finger force tasks (Fig. 4). For the extrinsic extensor muscle (Fig. 4A), the correlation coefficient (co-activation level) of all the affected fingers of stroke survivors were significantly higher than the control group ($p < 0.05$). We also found a significantly higher correlation coefficient in the affected fingers (index, ring, and little) than the contralateral fingers of stroke survivors ($p < 0.05$). In addition, we observed a higher correlation in the contralateral fingers (index, ring, and little) of stroke survivors

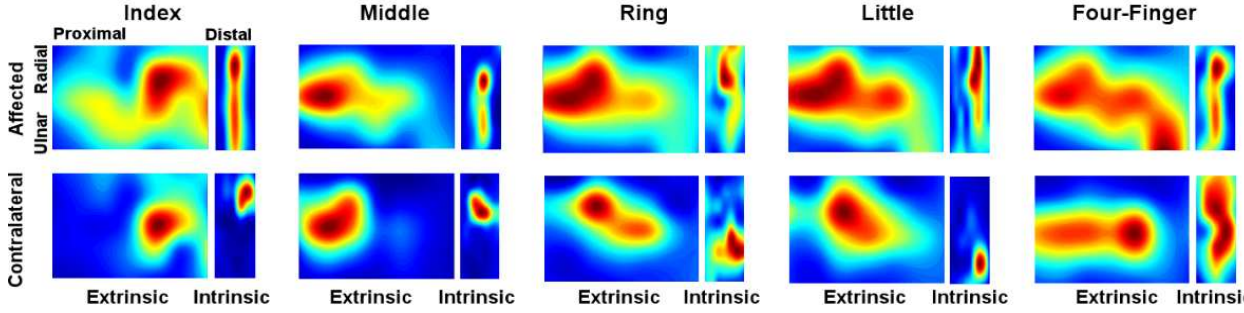

Fig. 3: Normalized extrinsic and intrinsic muscle activation from a stroke subject during single-finger and four-finger extensions. Warmer color indicates higher EMG energy.

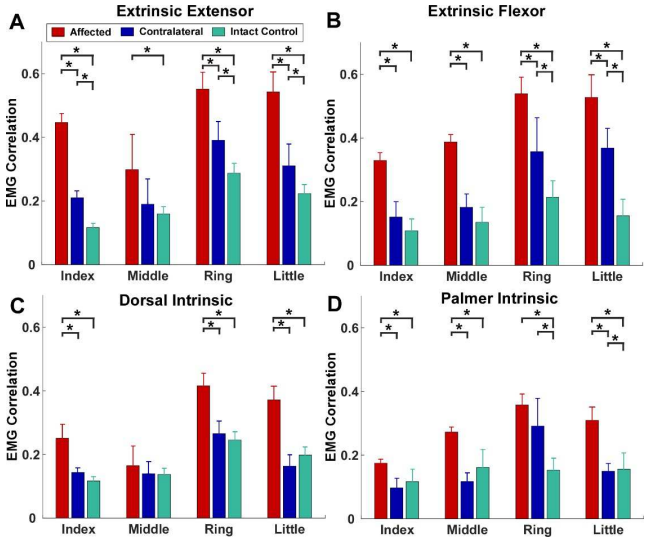

Fig. 4: Muscle co-activation quantified by EMG 2D correlation between single-finger and four-finger tasks. (A): EMG correlation coefficient of the extrinsic extensor muscle of the affected and contralateral sides of stroke survivors and average of both sides of the controls. (B): EMG correlation coefficient of the extrinsic flexor muscle. (C): EMG correlation coefficient of the dorsal intrinsic muscle. (D): EMG correlation coefficient of the palmer intrinsic muscle.

than that of the control group ($p < 0.05$). For the extrinsic flexor muscle (Fig. 4B), the correlation coefficient of all the affected fingers of stroke survivors were significantly higher than the contralateral side of stroke survivors and the control group ($p < 0.05$). In addition, we found a higher correlation in the contralateral ring and little fingers of stroke survivors than that of the control group ($p < 0.05$). For the dorsal intrinsic muscle (Fig. 4C), the correlation coefficient of the affected fingers (index, ring, and little) of stroke survivors were significantly higher than the contralateral side of stroke survivors and the control group ($p < 0.05$). For the palmer intrinsic muscle (Fig. 4D), the correlation coefficient of all the affected fingers of stroke survivors were significantly higher than the control group ($p < 0.05$). We also found a significantly higher correlation coefficient in the affected fingers (index, middle, and little) than the contralateral fingers of stroke survivors ($p < 0.05$). In addition, we found a higher correlation in the contralateral ring and little fingers of stroke survivors than that of the control group ($p < 0.05$). Lastly, we observed higher correlation coefficients in the extrinsic muscles than that of the intrinsic muscles ($p < 0.05$).

B. Finger Force Independence

We quantified the degree of independent finger force output by calculating the dimensionality (PCA) of the joint extension/flexion forces. As shown in Fig. 5, four PCs were required to capture the majority of the variance in the finger forces of the contralateral hand. In contrast, a single PC was sufficient to capture the majority of the force variance in the affected hand. The average difference of variance accounted for between the first PC and the remaining three PCs was calculated as an index of finger independence. A higher difference indicates less finger independence (more deficits).

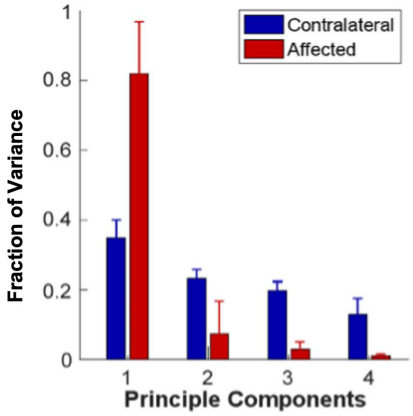

Fig. 5: The principal components (PC) in descending order of individual finger forces. Four PCs are needed to capture 95% of variance in the contralateral hand, and a single PC can capture 95% of variance in the affected hand.

Fig. 6 illustrates the muscle co-activation (EMG correlation coefficient) in association with the finger independence (difference of variance between the first and the remaining PCs). The results revealed that abnormal muscle co-activation in both flexors and extensors, especially the extrinsic muscles ($r = 0.69$ for extrinsic extensor and $r = 0.73$ for extrinsic flexor), tend to exhibit a higher association with finger force independence compared with the intrinsic finger muscles ($r = 0.48$ for dorsal intrinsic and $r = 0.52$ for palmer intrinsic). These findings suggest that excessive extrinsic muscle co-activation plays a greater role in impairment of finger independence than the intrinsic muscles.

We also quantified the association between abnormal muscle co-activation (EMG correlation asymmetry) and clinical assessment scales (Fig. 7). Our derived abnormal muscle co-activation index showed high correlation with ARAT ($r = 0.83$) and Chedoke scores ($r = 0.78$), indicating an effective metric to quantify hand impairment of stroke survivors.

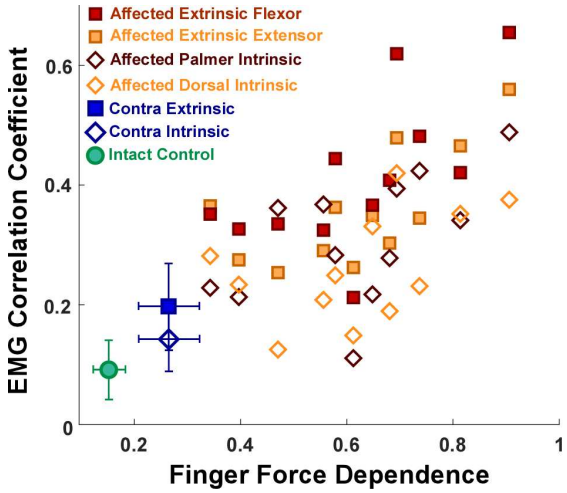

Fig. 6: Association between muscle co-activation (EMG correlation coefficient) and finger independence. All the contralateral extrinsic and intrinsic finger muscles and the control group were averaged respectively with error bars representing standard errors.

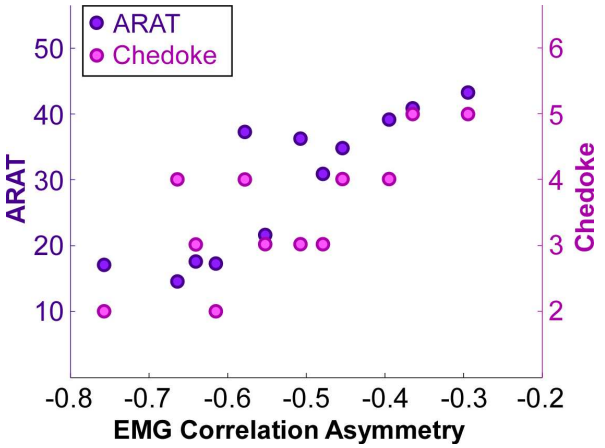

Fig. 7: Association between abnormal muscle co-activation (EMG correlation asymmetry) and clinical assessment scales.

C. Motor Unit Action Potential Distribution

We analyzed the spatial distribution of motor unit action potentials to identify potential spinal motoneuron reinnervation of the extrinsic muscle. To quantify the spatial spread of the action potentials of individual motor units, we calculated the SD of the active channel spatial locations of individual finger muscles (Fig. 8A). The results showed that there was a significant increase in the spread of action potentials for the index, middle, and ring & little fingers of the affected side in comparison with the contralateral side of stroke survivors and control subjects ($p < 0.05$). We merged the ring & little fingers because the spatial pattern of muscle activation and action potential distribution largely overlaps between these fingers [24]–[26]. We also quantified the association between asymmetry of channel variability and clinical scales (Fig. 8B). However, we only found weak correlations between these variables (ARAT: $r = 0.24$) and Chedoke: $r = 0.27$).

IV. Discussion

Besides muscular weakness and spasticity, a common manifestation of hand impairment following a stroke is the inability to control finger independently due to abnormal muscle co-activation patterns. In this study, we quantified the degree of muscle co-activation at the macro- and micro-levels based on HD-sEMG signals from both extrinsic and intrinsic muscle groups. We compared the macro-level muscle co-activation patterns of the affected and contralateral sides of stroke survivors, alongside the neurologically intact controls. Our results revealed a significantly higher co-activation level in the affected extrinsic muscles and to a lesser degree in the affected intrinsic muscles of stroke survivors, in comparison with the contralateral side and the control group. Moreover, the asymmetry in muscle co-activation exhibited a strong correlation with impaired finger independence and clinical assessment scales (ARAT and Chedoke-McMaster Assessment). Through the micro-level analysis of MUAP distribution, we observed a significant increase in the spread of action potentials for individual extrinsic muscles on the affected side. However, the altered MUAP distribution did not correlate with clinical assessment scales. Collectively, we identify the abnormalities of muscle co-activation that can contribute to impaired finger independence. Our research outcomes provide a systematic understanding of the pathophysiology of hand dexterity deficits of stroke survivors. This understanding can provide a theoretical basis for the development of early intervention strategies that can potentially reduce or even prevent these maladaptive changes after the initial lesion.

A. The Comparison between Extrinsic and Intrinsic Muscles

The examination of extrinsic and intrinsic muscles in the context of post-stroke hand impairment has been relatively imbalanced in the literature, with a predominant focus on extrinsic muscles while paying less attention to the co-activation of intrinsic muscles [14][25][26]. Our study sought to rectify this by delving into the activation patterns of both muscle groups. The results revealed notably higher degrees of co-activation within the extrinsic muscle compartments compared to the intrinsic muscles on the affected sides of stroke survivors. Additionally, the co-activation of extrinsic muscles exhibited a stronger association with finger independence in contrast to the intrinsic muscles. Our results

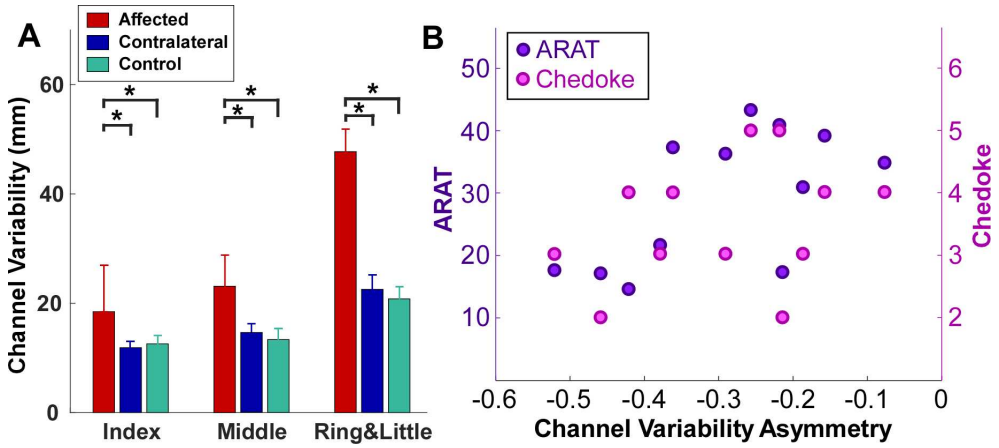

Fig. 8: Motor unit action potential distribution and clinical association. (A): The channel variability of individual finger muscles of the affected and contralateral sides of stroke survivors and average of both sides of the controls. (B): Association between MUAP distribution (channel variability asymmetry) and clinical assessment scales.

suggest that excessive co-activation in the extrinsic muscles plays a more substantial role in impairing finger independence than their intrinsic counterparts.

This observed phenomenon can be attributed to several potential reasons. Firstly, the extrinsic muscle group encompasses multiple multi-compartments and multi-tendonous muscles that control finger movements [29]. For instance, the extensor digitorum communis (EDC) is a multifaceted muscle crucial for the extension of four digits. Its compartments intricately manage the extension of individual fingers. After a stroke, maladaptive changes in cortical activation and damaged corticospinal projections can amplify shared inputs among these components, leading to the observed abnormal co-activation. Previous studies on motor unit synchronization have shown that there is a higher degree of independence in the intrinsic and the extrinsic muscles [30], [31] in intact controls. In addition, there is also evidence that impairment (such as muscle atrophy) is more severe in the extrinsic muscles than the intrinsic muscles post stroke [32], [33]. Secondly, the differential findings between extrinsic and intrinsic muscles may also be due to anatomical and physiological differences between these muscles [34]. The different intrinsic muscles are organized mechanically more independently compared with extrinsic muscles. The level of shared neural input among intrinsic muscles is weak relative to extrinsic compartments [35], [36]. Lastly, considering anatomical factors, extrinsic muscle compartments partially overlap, are organized obliquely, and are located at different depths relative to the skin surface [37], [38], posing challenges in isolating them using skin surface EMG signals. Future work using ultrasound-based muscle deformation [39] or HD-sEMG-based source localization approaches [40] to capture activation of individual muscle compartments can help address these challenges.

B. The Comparison between Contralateral Side and Controls

When quantifying motor impairment in stroke survivors, the affected side garner primary attention. The contralateral side always serves as control references, presumed to mirror intact controls [25][29]. However, our results show that the muscle activation patterns in the contralateral side also demonstrated abnormalities compared with intact controls. In our analysis of muscle co-activation, the muscle co-activation on the contralateral sides of stroke survivors were notably higher than those observed in intact controls in the extrinsic extensor compartments (index, ring, and little fingers) and extrinsic flexor compartments (ring and little fingers).

After a hemispheric stroke, the lesion can induce interhemispheric imbalance involving hyperexcitability of the contralesional hemisphere, and lesion-responsive reorganizations may occur on both hemispheres. The hyperexcitable contralesional hemisphere can contribute to increased muscle co-activations in both extrinsic extensor and flexor muscles as well as the intrinsic flexors. Our results extend previous observations of motor deficits in the ipsilesional side of stroke survivors [41]–[44]. Besides adaptations in the contralesional hemisphere, both hemispheres naturally contribute to unilateral hand motor functions [41], which can also lead to ipsilesional motor deficits.

C. The MUAP Distribution Changes at the Micro Level

Alongside examining muscle co-activation patterns at a macro level, our study delved into the micro-level analysis of MUAP distribution. The results revealed a significant increase in the spatial spread of action potentials on the affected side compared to both the contralateral side of stroke survivors and intact controls. These micro-level changes likely contribute to the observed abnormal muscle co-activation pattern evident in the macro-EMG signals. The observed changes in action potential distributions reflect signs of motoneuron loss and subsequent reinnervations of muscle fibers. There is evidence that neuronal reinnervation and motor unit re-distribution can occur in intrinsic finger muscles of chronic stroke survivors [46], partly due to motoneuron death and muscle fiber atrophy or loss [47]–[49]. The reinnervation process is typically not well organized, such that different fiber types can be reinnervated, leading to polyphasic action potentials and altered contractile properties of the motor units[50], [51]. Our findings provide knowledge regarding the extent of reinnervation and altered MUAP distribution across compartments of the extrinsic muscles. If their restructuring is similarly disorganized, this could contribute to increased coupling across fingers.

However, our investigation found a weak association between MUAP distribution and clinical scales (Fig. 8B). While these micro-level changes may play a role in muscle co-activation, they do not seem to reliably contribute to hand functional impairments in stroke survivors. Instead, the asymmetry in EMG correlation, which reflects muscle co-activation patterns at the macro level, emerges as a more accurate and robust assessment tool for evaluating hand functional impairments.

D. Limitations

As outlined in the Introduction section, standardized clinical assessments inherently possess subjectivity and intermittency. The ARAT and Chedoke scores acquired from a single test session for our stroke subjects might not offer an entirely objective and accurate measurement of hand function impairments. The inherent bias within these clinical scores could potentially limit the correlation observed between abnormal muscle co-activation and clinical assessment scales.

Our findings show a correlation between HD-sEMG metrics and clinical assessments, but only in chronic (>6 months post stroke) stroke survivors, limiting our data's diversity. An ongoing study on subacute stroke survivors aims to compare their metrics with those of chronic patients. We also plan to expand our sample size to evaluate the feasibility of these outcome measures more comprehensively for clinical use.

Our study was conducted in a controlled laboratory setting with complex data post-processing procedures, currently unsuitable for direct home use. While HD-sEMG metrics show promise as objective measures compared to clinical scales, future home deployment requires further advancements in

wearable sensing and computing technologies. This would allow HD-sEMG recordings to be obtained from a fully wearable system, and derived metrics to be calculated automatically, without user interactions.

V. Conclusion

Overall, using HD-sEMG recording arrays and specialized signal processing techniques, we systematically quantified activation patterns of extrinsic and intrinsic finger muscles in stroke survivors and intact controls. The results revealed that abnormal co-activation correlated with finger independence and clinical assessment scales. We also found that altered MUAP distribution was evident in extrinsic finger muscles, which may partly contribute to the abnormal muscle co-activation patterns. Our work can provide a better understanding of the mechanisms of impaired finger independence and provide a novel perspective for hand function assessment in stroke survivors.

References

[1] C. W. Tsao *et al.*, "Heart disease and stroke statistics—2023 update: A report from the American heart association," *Circulation*, vol. 147, no. 8, pp. e93–e621, Feb. 2023.

[2] C. D. A. Wolfe et al., "The impact of stroke." [Online]. Available: https://academic.oup.com/bmb/article/56/2/275/303250

[3] K. Sathian *et al.*, "Neurological principles and rehabilitation of action disorders: common clinical deficits," *Neurorehabil Neural Repair*, vol. 25, pp. 21S-32S, 2011.

[4] A. Sunderland et al., "Arm function after stroke. An evaluation of grip strength as a measure of recovery and a prognostic indicator," *J Neurol Neurosurg Psychiatry*, vol. 52, no. 11, pp. 1267–1272, 1989.

[5] D. T. Wade et al., "The hemiplegic arm after stroke: Measurement and recovery," *J Neurol Neurosurg Psychiatry*, vol. 46, no. 6, pp. 521–524, 1983.

[6] J. P. A. Dewald et al., "Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects," 1995.

[7] C. E. Lang and M. H. Schieber, "Differential impairment of individuated finger movements in humans after damage to the motor cortex or the corticospinal tract," *J Neurophysiol*, vol. 90, no. 2, pp. 1160–1170, Aug. 2003.

[8] C. E. Lang and M. H. Schieber, "Reduced muscle selectivity during individuated finger movements in humans after damage to the motor cortex or corticospinal tract," *J Neurophysiol*, vol. 91, no. 4, pp. 1722–1733, Apr. 2004.

[9] J. N. A. L. Leijnse et al., "Assessment of individual finger muscle activity in the extensor digitorum communis by surface EMG," *J Neurophysiol*, vol. 100, no. 6, pp. 3225–3235, Dec. 2008.

[10] C. E. Lang and M. H. Schieber, "Reduced muscle selectivity during individuated finger movements in humans after damage to the motor cortex or corticospinal tract," *J Neurophysiol*, vol. 91, no. 4, pp. 1722–1733, Apr. 2004.

[11] C. E. Lang et al., "Assessment of upper extremity impairment, function, and activity after stroke: Foundations for clinical decision making," *Journal of Hand Therapy*, vol. 26, no. 2, pp. 104–115, Apr. 2013.

[12] N. Yozbatiran et al., "A standardized approach to performing the action research arm test," *Neurorehabil Neural Repair*, vol. 22, no. 1, pp. 78–90, Jan. 2008.

[13] C. Gowland *et al.*, "Measuring physical impairment and disability with the Chedoke-McMaster stroke assessment." [Online]. Available: http://ahajournals.org

[14] J. Fan et al., "Assessment of impaired finger independence of stroke survivors: A preliminary study," in *International IEEE/EMBS Conference on Neural Engineering, NER*, IEEE Computer Society, 2023.

[15] Z. M. Lí Latash and V. Zatsiorsky, "Force sharing among fingers as a model of the redundancy problem," Springer-Verlag, 1998.

[16] Z. M. Li, V. M. Zatsiorsky et al., "Bilateral multifinger deficits in symmetric key-pressing tasks," *Exp Brain Res*, vol. 140, no. 1, pp. 86–94, 2001.

[17] Y. Zheng and X. Hu, "Interference removal from electromyography based on independent component analysis," *IEEE Transactions on Neural Systems and Rehabilitation Engineering*, vol. 27, no. 5, pp. 887–894, May 2019.

[18] M. Santello et al., "Neural bases of hand synergies," *Front Comput Neurosci*, no. MAR, Mar. 2013.

[19] C. Dai and X. Hu, "Independent component analysis based algorithms for high-density electromyogram decomposition: systematic evaluation through simulation," 2019.

[20] C. Dai and X. Hu, "Independent component analysis based algorithms for high-density electromyogram decomposition: experimental evaluation of upper extremity muscles," 2019.

[21] C. Dai et al., "Prediction of individual finger forces based on decoded motoneuron activities," *Ann Biomed Eng*, vol. 47, no. 6, pp. 1357–1368, Jun. 2019.

[22] X. Hu et al., "Motor unit pool organization examined via spike-triggered averaging of the surface electromyogram," *J Neurophysiol*, vol. 110, no. 5, pp. 1205–1220, Sep. 2013.

[23] X. Hu et al., "Reliability of spike triggered averaging of the surface electromyogram for motor unit action potential estimation," *Muscle Nerve*, vol. 48, no. 4, pp. 557–570, 2013.

[24] C. Dai and X. Hu, "Extracting and classifying spatial muscle activation patterns in forearm flexor muscles using high-density electromyogram recordings," *Int J Neural Syst*, vol. 29, no. 1, Feb. 2019.

[25] X. Hu et al., "Extracting extensor digitorum communis activation patterns using high-density surface electromyography," *Front Physiol*, vol. 6, Oct, 2015.

[26] C. Dai et al., "Estimation of muscle force based on neural drive in a hemispheric stroke survivor," *Front Neurol*, vol. 9, no. MAR, Mar. 2018.

[27] W. Sheng *et al.*, "Upper limbs muscle co-contraction changes correlated with the impairment of the corticospinal tract in stroke survivors: Preliminary evidence from electromyography and motor-evoked potential," *Front Neurosci*, vol. 16, Jun. 2022.

[28] S. W. Lee, K. Triandafilou et al., "Impairment in task-specific modulation of muscle coordination correlates with the severity of hand impairment following stroke," *PLoS One*, vol. 8, no. 7, Jul. 2013.

[29] S. Standring, *Gray's Anatomy*, 40th ed. New York, USA: Churchill Livingstone, 2008.

[30] SA. Winges et al., "Common input to motor units of intrinsic and extrinsic hand muscles during two-digit object hold," *J Neurophysiol*, vol. 99, no. 3, Mar. 2008.

[31] AJ. Fuglevand, "Mechanical properties and neural control of human hand motor units," *J Physiol.,* vol. 589, no. Pt23, pp. 5595-5602, Dec. 2011.

[32] KM. Triandafilou et al., "Investigation of hand muscle atrophy in stroke survivors," *Clin Biomech (Bristol, Avon)*, vol. 27, no. 3, pp. 268-272, Mar. 2012.

[33] EG. Cruz et al., "Kinetic and kinematic workspaces of the index finger following stroke," *Brain*, vol. 128, no. Pt 5, pp.1112-1121, May 2005.

[34] M. H. Schieber and M. Santello, "Hand function: peripheral and central constraints on performance," *J Appl Physiol*, vol. 96, pp. 2293–2300, 2004.

[35] M. Santello and A. J. Fuglevand, "Role of across-muscle motor unit synchrony for the coordination of forces," *Exp Brain Res*, vol. 159, no. 4, pp. 501–508, Dec. 2004.

[36] S. A. Winges et al., "Common input to motor units of intrinsic and extrinsic hand muscles during two-digit object hold," *J Neurophysiol*, vol. 99, no. 3, pp. 1119–1126, Mar. 2008.

[37] J. N. A. L. Leijnse et al., "Assessment of individual finger muscle activity in the extensor digitorum communis by surface EMG," *J Neurophysiol*, vol. 100, no. 6, pp. 3225–3235, Dec. 2008.

[38] J. N. A. L. Leijnse et al., "Anatomic basis for individuated surface EMG and homogeneous electrostimulation with neuroprostheses of the extensor digitorum communis," *J Neurophysiol*, vol. 100, no. 1, pp. 64–75, Jul. 2008.

[39] Y. Zheng et al., "Automatic detection of contracting muscle regions via the deformation field of transverse ultrasound images: A feasibility study," *Ann Biomed Eng*, vol. 49, no. 1, pp. 354–366, Jan. 2021.

[40] Y. Liu et al., "Motor unit distribution and recruitment in spastic and non-spastic bilateral biceps brachii muscles of chronic stroke survivors," *J Neural Eng*, vol. 19, no. 4, Aug. 2022.

[41] C. Dai et al., "Altered motor unit discharge coherence in paretic muscles of stroke survivors," *Front Neurol*, vol. 8, no. MAY, May 2017.

[42] C. Chestnut and K. Y. Haaland, "Functional significance of ipsilesional motor deficits after unilateral stroke," *Arch Phys Med Rehabil*, vol. 89, no. 1, pp. 62–68, Jan. 2008.
[43] C. J. Winstein et al., "Effects of unilateral brain damage on the control of goal-directed hand movements," Springer-Verlag, 1995.
[44] J. R. Carey et al., "Tracking control in the nonparetic hand of subjects with stroke," 1998.
[45] S. Y. Schaefer et al., "Ipsilesional motor deficits following stroke reflect hemispheric specializations for movement control," *Brain*, vol. 130, no. 8, pp. 2146–2158, Aug. 2007.
[46] B. Jeon et al., "Motor unit structural change post stroke examined via surface electromyography: a preliminary report," in *2013 6th International Ieee/Embs Conference on Neural Engineering (Ner)*, 2013, pp. 1234–1237.
[47] J. J. Gemperline et al., "Characteristics of motor unit discharge in subjects with hemiparesis," *Muscle Nerve*, vol. 18, no. 10, pp. 1101–1114, Oct. 1995.
[48] S. Chokroverty et al., "Hemiplegic amyotrophy muscle and motor point biopsy study," *Arch Neurol*, vol. 33, no. 2, pp. 104–110, Feb. 1976, [Online]. Available: http://archneur.jamanetwork.com/
[49] X. Li et al., "Motor unit number reductions in paretic muscles of stroke survivors," *IEEE Transactions on Information Technology in Biomedicine*, vol. 15, no. 4, pp. 505–512, Jul. 2011.
[50] A. Cruz Martinez et al., "Altered motor unit architecture in hemiparetic patients. A single fibre EMG study," *J Neurol Neurosurg Psychiatry*, vol. 45, no. 8, pp. 756–757, Aug. 1982.
[51] M. Lukács, "Electrophysiological signs of changes in motor units after ischaemic stroke," *Clinical Neurophysiology*, vol. 116, no. 7, pp. 1566–1570, Jul. 2005.